Hunting for hidden hypertensive etiologies

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Story from the front lines

A man in his 70s with congestive heart failure, coronary artery disease, and chronic kidney disease underwent an outpatient workup for worsening hypertension. He had a 25-year history of hypertension with systolic blood pressure trends in the 130s-140s; however in the past two years his systolic blood pressure began to climb into the 140s-160s, prompting uptitration of his antihypertensive regimen that consisted of hydrochlorothiazide, losartan, and metoprolol.

He was evaluated for secondary causes of hypertension with a workup that included urine and plasma metanephrines, thyroid studies, plasma renin, and aldosterone, all of which returned within normal limits. A renal ultrasound was obtained and demonstrated normal arterial flow. At this point he was thought to have either worsening essential hypertension due to aging or secondary hypertension from a renovascular cause. He was then sent to have a renal MRA with contrast to rule out renal artery stenosis (RAS); however due to his body habitus he was unable to undergo the scan. Ultimately it was felt that obtaining the MRA was unnecessary, and the patient's hypertension was medically managed by optimizing his antihypertensive regimen.

Teachable moment

There are several instructive points for further discussion that could be extracted from this case, such as appropriate goals for blood pressure treatment in this patient; the consideration between ultrasound and MRA in terms of sensitivity, specificity, costs, and complications; or the utility of searching for endocrine and other less common etiologies of secondary hypertension in a patient with several more common reasons for elevated blood pressure (e.g., kidney disease, sleep apnea, and normal aging). The focus of this discussion will be on establishing the diagnosis of RAS and the implications of such.

Atherosclerotic RAS is a likely differential diagnosis for this patient given his kidney disease and known atherosclerosis in his coronary and carotid arteries. It is one of the most common causes of secondary hypertension, occurring in an estimated 7% of elderly patients, and is associated with an increased risk of cardiovascular events such as stroke and decreased survival compared to patients without RAS. Often clinical clues such as refractory hypertension or comorbid renal insufficiency are suggestive of RAS, but the diagnosis is made definitively with imaging.[1]

While it may have been reassuring to both patient and physician to have a conclusive answer to rising blood pressure, there was little benefit in obtaining further imaging, especially after an ultrasound was negative for RAS. Certainly there is risk of harm induced by MRA itself. Gadolinium, for example, has been associated with adverse reactions, including nephrogenic systemic fibrosis, especially in patients with renal disease.[2] More significantly, however, there is a low likelihood that the results of additional imaging would change the management of this patient's blood pressure.

According to current guidelines, undergoing diagnostic studies to evaluate for RAS is indicated only in cases where percutaneous or surgical revascularization would be pursued if RAS were diagnosed.[3] In our patient's case, given his comorbidities he was a poor candidate for any procedural intervention had he been found to have confirmed RAS. More importantly, the risks of revascularization would have outweighed any foreseeable benefits, as studies have established that for patients with RAS, there are no benefits to management with renal artery vascularization compared to management with medical therapy alone, with regards to blood pressure, [4,5] stabilization of CKD, or long-term clinical outcomes.[6] Moreover it is uncommon for patients who are treated with medical therapy alone to progress to end-stage renal disease or to develop refractory hypertension.[1] Had our patient been able to obtain the renal MRA, he would nevertheless have been medically managed with antihypertensive optimization and risk factor reduction regardless of the imaging results.

In the hunt for a clinical answer, it can be challenging to leave a diagnosis unconfirmed when there is an abundance of diagnostic tools readily available. Our patient's inadvertent cancellation of his MRA fortuitously gave pause for consideration of the necessity of the test. Establishing the diagnosis of RAS may have been reassuring but was very unlikely to change management, and the hunt was thereby concluded.

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